cases. Second, important cellular events that cause secondary injury may be similar or even identical in both stroke and trauma. The animal models described by the authors, employing sequential injury—impact followed by ischemia—are, then, appropriate from both perspectives. Further, these models at least indirectly address the central hypothesis of increased neuronal vulnerability and secondary cell death after injury. They also define specific candidate mechanisms that may underlie this vulnerability and provide new directions for therapy.

Several published reports have demonstrated the coincidence of head injury and shock or hypoxia. For example, consider data from the pilot phase of the national Traumatic Coma Data Bank.[1] In that study, data were collected prospectively on almost 600 patients. Of these patients, 20% were considered hypoxic before admission, as defined by an initial partial arterial oxygen pressure of 60 torr or less, or by a history of apnea in those patients ventilated before their arterial blood gases were measured. Shock occurred in 33% of the population as defined by a systolic blood pressure of 90 torr or less before admission. Shock was associated with a 30% increase in the frequency of the outcome of brain death or vegetative state, while hypoxia was associated with about a 20% increase in this outcome. Additionally, in association with these insults there were relatively fewer patients who were considered to have a good outcome or to be only moderately disabled. These associations were statistically significant. Further, while there is, no doubt, an association between multisystem injury and either shock or hypoxia, a statistically significant association between multisystem trauma and outcome could not be found in this population. Data from the full-phase study, which includes information from more than 1,000 other patients, are currently undergoing analysis and also show the importance of early hypoxia and shock in patients with severe head injury.

What are the specific cellular mechanisms common to stroke and trauma that could cause increased neuronal vulnerability in secondary cell death? Those currently under the most intensive investigation and discussed by Becker and co-workers have a central theme of the release of substances from injured or dying cells, substances assumed toxic to nearby neurons spared by the initial precipitating event. These mechanisms include the accumulation of lactate and acidification of the local environment; cell-membrane breakdown, lipid peroxidation, and release of free radicals; and excessive release and accumulation of neurotoxins and excitatory synaptic transmitters, particularly glutamate. A recently published report from the Cornell group headed by Plum and Pulsinelli has provided important direct evidence that lactate in concentrations like those found after ischemia can cause neuronal death.[2] These investigators studied the histologic appearance of rat brains that had been subjected to microinjections of various concentrations of lactate. The idea that lactate or hydrogen ion is toxic to neurons now seems secure. In view of the controversies surrounding this idea, it is surprising that this experiment was not done earlier. Also recently reported and interesting are the findings of Pitts and associates.[3] They studied a sequential injury model using magnetic resonance spectroscopy. Pertinent to the hypothesis under consideration here was the finding that intracellular pH and high-energy phosphate concentrations (adenosine triphosphate and creatine phosphate) decreased when both injuries were combined—impact followed by hypoxia—to a much greater degree than when the two injuries were studied separately.

While the idea that glutamate in high concentrations may act as a neurotoxin is not new, compounds that would easily penetrate the blood-brain barrier and act as specific receptor (*N*-methyl-D-aspartate) antagonists have only recently become available, and one such compound, MK-801, has been shown in animal models of ischemia to have beneficial effects.[4] Of the three outlined mechanisms, the release of free radicals as a cause of secondary neuronal injury in animal models has been the most widely supported; data have been generated from many laboratories under a wide variety of conditions. The evidence is sufficiently impressive to warrant a clinical trial in the context of a multicenter study, and the United States Department of Defense has funded such a trial of superoxide dismutase in patients with missile injuries of the brain.

There has been considerable progress in studying these mechanisms, though their clinical importance must await further study. It is hoped that when indicated, they will be tested by multicenter, randomized trials. The clinical importance of cellular mechanisms in the animal studies of long-term recovery, particularly those indicating a possible role for axonal sprouting, will obviously be more difficult to evaluate. These findings are potentially the most exciting, however, and these kinds of experiments should be encouraged.

HOWARD M. EISENBERG, MD
*Division of Neurosurgery*
*The University of Texas Medical Branch*
*Galveston, Texas*

**REFERENCES**

1. Eisenberg HM, Cayard C, Papanicolaou AC, et al: The effects of three potentially preventable complications on outcome after severe head injury, chap 93, *In* Heidelberg V (Ed): Intracranial Pressure. New York, Springer-Verlag, 1983, pp 549-553

2. Kraig RP, Petito CK, Plum F, et al: Hydrogen ions kill brain at concentrations reached in ischemia. J Cereb Blood Flow Metab 1987; 7:379-386

3. Ishige N, Pitts LH, Pogliani L, et al: Effect of hypoxia on traumatic brain injury in rats: Pt 2. Changes in high energy phosphate metabolism. Neurosurgery 1987; 20:854-858

4. Kochhar A, Zivin JA, Lyden PD, et al: Glutamate antagonist therapy reduces neurologic deficits produced by focal central nervous system ischemia. Arch Neurol 1988; 45:148-153

## Uncompensated Care

"MY JOB AS A PHYSICIAN is to serve as an agent of trust and to do anything appropriate to provide necessary medical care and not to serve as a rationeer of scarce resources on an individual basis." This statement of Dr Nancy Dickey, chair of the American Medical Association's Council on Ethical and Judicial Affairs, expressed the convictions of most physicians. None of us, as physicians and human beings, want to make our professional decisions for our patients primarily for fiscal reasons. We desire to give them quality care and to be their advocates when needed.

Society may change this. It may mandate us to render inadequate or improper services if the problems associated with uncompensated care are not solved. The growing crisis of caring for those who are uninsured, underinsured, and uninsurable, according to Dr John Kitzhaber, ". . . poses one of the most serious threats facing the medical profession today." It can result in a deterioration of health for a growing number of Americans, with very serious social and economic consequences.

Dr Kitzhaber, whose address to the California Medical Association House of Delegates at its 1988 annual meeting is

published elsewhere in this issue, is a practicing physician in Roseburg, Oregon, and a state legislator. At age 41, he is president of the Oregon State Senate and an expert dealing with a variety of issues including public education, land use planning, water policy, and health care. His discussion of the problem of uncompensated care—its etiology, its symptomatology, its prognosis, and its possible cure—reveals the depth of his concern and expertise.

The problem of uncompensated care—giving services to those with no means to pay—is not new in America. It has been present since colonial days, and yet, the poor have always received a modicum of health care. Some came from charity but most, because of the influence of the Elizabethan poor laws, came from local political subdivisions: towns, cities, or counties. Both the facilities and the care were primitive, barely giving enough sustenance to sustain life. Gradually, the poor farms and pesthouses of the past century gave way to county hospitals, some of which became the great teaching institutions of today affiliated with medical schools. Before the 1960s the poor had easy access to these institutions. Although the accommodations on bleak and crowded wards were usually spartan, the quality of both medical and nursing care often equalled or exceeded that given in private institutions. Most of the nonprofit and religion-controlled hospitals gave a substantial number of people care at reduced rates. The fee-for-service system in existence then allowed and even encouraged providers—both physicians and hospitals—to shift costs. It permitted them to charge the wealthy more in order to pay for the care of the poor.

The principle of universal access mentioned by Dr Kitzhaber, "the idea that all Americans, regardless of their income, should have access to the health care system and to all the services it had to offer," came into full fruition during the 1960s with the enactment of Medicare and Medicaid and the growth of "private health insurance policies funded primarily through employment."

For a while this system worked quite well. America seemed to have achieved "an ideal health system." Almost everyone had access to mainline quality care: the elderly were covered by Medicare, the workers and their families by employer-funded insurance, and the poor by Medicaid. This egalitarianism of care prompted third-party payers to pay a consistent and uniform fee to each provider. This seemed fair, but over the years it effectively stopped cost shifting.

Meanwhile, the overall cost of health care escalated in two decades from 7% of the gross national product to the current 11%. This resulted from many factors, including a growing population, especially of older people, increased technology, inflation, rising expectations, and growing demands for more services. As the costs of care increased, the prosperity of the country declined, the national debt increased, workers' productivity decreased, and American businesses could no longer compete with foreign manufacturers, either abroad or at home. Neither the public nor the private sector could afford the high cost of medical care. Both reacted by cutting back their coverage. This widened the gap between the two, leaving more people without health insurance. Since these people have limited fiscal resources, they can either go without medical services or seek uncompensated care. Since shifting of costs is no longer allowed, providers are reluctant to care for this growing segment of the population.

To date, little has been done to resolve the problem except to lay blame on providers. Since physicians are perceived to be wealthy, many members of society feel that doctors should be mandated to take charity cases. In fact, some legislatures are considering making this a requirement for continued licensure. Because many nonprofit hospitals received federal funds in the past, they had an obligation to care for uncompensated patients. More and more nonprofit hospitals, however, are closing their doors because of financial difficulties. This compounds the problem. The absence of cost shifting makes the providers—physicians and hospitals—face the prospect of caring for more patients without adequate recompense, refusing them care, or sending them elsewhere: "dumping."

Neither of these alternatives leads to meaningful care for these unfortunate people. Dr Kitzhaber believes society must make the hard decisions needed to solve the problem of uncompensated care by creating a new system of health care based on limited resources and acceptance of the fact that the well-off can purchase more health care than the poor—the reality of implicit rationing. "The government," he states, "should pay for the poor regardless of their age" but not for the elderly. The new health care system that Dr Kitzhaber recommends would have a minimum of three tiers: government-sponsored for the poor; employer-funded for the workers; and a traditional fee-for-service tier for those who wish to buy the type of health care they desire.

As this concept develops, the medical profession has the responsibility of defining the various levels of care, especially the basic and minimal level available to the poor. This means that the emphasis must be placed on what is best for society, not what is best for the individual. Once the basic level of health care is defined, society has no recourse except to pay for it through government funding. If society in its collective wisdom feels that additional services are needed for the poor, it has the option to provide them based upon the amount of money it wants to spend.

In a system such as this, the money spent on health care for the poor will be used most effectively for the good of society. Working with these patients, physicians know the limitations of their service; they are not being put in the role of rationeer. If, according to Dr Kitzhaber, they feel their patients need more than the government allows, they can truly be advocates by appealing on their patients' behalf. This would allow physicians to continue to serve as agents of trust and to do anything appropriate to provide necessary medical care.

RICHARD L. JOHNSON, MD
*Sacramento, California*

## Suction-Assisted Lipectomy—Caveat Emptor

THE ARTICLE BY BELLO and co-workers elsewhere in this issue adds to our knowledge and awareness that severe infections may ensue following suction-assisted lipectomy. The medical profession was warned of this possibility in 1982 by the ad hoc committee that was formed by the American Society of Plastic and Reconstructive Surgeons to study the subject. Five years later and with an experience of more than 100,000 cases, the society reported to the profession and the public at large a total of 11 deaths and 9 cases of serious life-threatening complications.[1] Several of the deaths were attributed to severe necrotizing fasciitis.